MENINGITIS OF NASAL ORIGIN.*

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The subject of meningitis of nasal origin is one with a compendious literature. In considering it in a paper of this scope we must confine ourselves to well ascertained facts, instructive and interesting as the many theories may be.

I wish briefly to call to mind a few peculiarities of the anatomy of this region, which would seem to make it an ideal starting point for the disease in question. The inspired air, carrying many pathogenic organisms, constantly passes through the nares. The various accessory cavities with their mucous membrane lining, small ostia, and favorable conditions of heat, moisture, and dead organic matter, would apparently offer an ideal cultural ground under local or systemic conditions of lowered resistance, were it not for certain protective agencies, viz: The expulsive action of the ciliated epithelium; a supposed bactericidal action of the nasal mucous,[1] its being a poor culture medium,[2] and its mechanical agglutination and enmeshing of the bacteria,[3] we would find meningitis from this source an extremely common thing. Another saving factor which may be mentioned is that the nasal mucous membrane has a collateral blood supply from the bone itself. There is also no doubt that an immunity to many organisms exists in the nose, owing to their constant presence there, and that this immunity is only overcome by fresh invasion of virulent germs, or lighting up of avirulent ones by systemic or local depression.

The frontal sinus and ethmoid labyrinth are in direct relation to the dura; the sphenoid to the cavernous sinus, the maxillary antrum to the orbit. The veins of the frontal sinus anastomose with the longitudinal sinus,[4] the veins of the ethmoid empty into the superior and sometimes the inferior ophthalmic vein, the veins of the ethmoid also anastomose with those of the dura, and the veins of the sphenoid anastomose with the cavernous sinus. Killian demonstrated communication between veins of sphenoid sinus and the sheath of the optic nerve by means of silver injections.[5] Here, then, in the venous anastomosis is one very apparent route of invasion. We have also, in not very infrequent instances, dehiscences of the walls of these cavities, thus bringing the nasal mucosa in direct contact with dura. The lymphatics of the nose have a direct connection with the peri-meninges.[6] The olfactory nerves communicate directly with the nasal mucosa through the cribiform plate.

The modes of invasion of the meninges are:

First: By sinus disease, either acure or chronic, causing necrosis of underlying bone. When there is a dehiscence in the bony wall the invasion is through mucous membrane direct. There may be an invasion through the bone, with no apparent involvement of it,[7] but Hajek and others have shown that there is a hemorrhagic infiltration as well as numerous bacteria present in the bone substance in such cases.[8]

Second: By thrombosis of brain sinuses by means of communicating venous channels. The sphenoid sinus is most commonly the source of thrombophlebitis complications, the ethmoid next through the anterior and posterior ethmoidal veins.

Third: By lymph channels. There is a considerable doubt as to these being instrumental in conveying the disease. Logan, Turner, Gerber, Hoffman, Ogston, Mayer and many others are of the opinion that they play little or no part in the process.

Fourth: By the fibres and sheaths of the olfactory nerves. This was proved in Hayen's case, in which he packed a nose with perchlorid gauze for bleeding. This patient died a few days later from meningitis and the autopsy showed the olfactory tract and nerve fibres stained brown to the meninges.

Fifth: By involvement of neighboring structures. Meningitis has been caused by maxillary sinus infection extending to the orbit, the abscess thus produced infecting the meninges by way of the optic foramen, or ophthalmic vein.

Sixth: By bacterial invasion of the blood current without thrombophlebitis. This is shown in cerebrospinal meningitis, where the meningococcus is harbored in the naso-pharynx and posterior nares. The germ is always recovered in the blood, and apparently reaches the brain covering in no other manner.[9]

These, then, constitute the avenues of infection of the meninges. The diagnosis, pathology and bacteriology of the disease are the same as in that arising from any other source, and will be omitted.

From the standpoint of origin, we may classify meningitis as:

First: Those cases arising from chronic sinus infections. These constitute the greatest number of cases of nasal origin apart from the epidemic form. The frontal sinus, owing to its large surface contact with the dura and its frequently poor drainage due to stenosis of the duct, internal septa, etc., is responsible for more cases than the other sinuses,[10] the sphenoid coming next.

Second: Those arising from acute infections.

Third: Those arising from trauma. Operative interference in the nose undoubtedly would cause a woeful number of cases were it not for the natural defenses provided in the nose and general system, which have been previously referred to. Careless and unskilful surgery have taken their toll of deaths from this affection. Nor is this to be wondered at, when one thinks of the comparatively thin shell of bone protecting the brain in this region, the opening up of the numerous channels of communication with the meninges, the possible breaking down of barriers around walled off infections and the introduction of fresh germs.

I have not mentioned at any length that form of meningitis produced by the meningococcus of Weishelbaum. This is not of such special interest to rhinologists as the other forms, for it produces very little pathology in the nose proper, which is mainly its culture field. It is of importance to us, however, in that we should be able to recognize it during epidemics and be prepared to guard

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against the spread of the disease by culturing all suspicious cases. As this is the only means so far as known of its spread, it assumes a place of first importance.[11]

Fortunately the meningococcus is short-lived outside of its natural habitat, the nasopharynx.

Peters [12] classifies cerebrospinal fever into two types: First, those in whom the sphenoid sinuses are patent; Second, those in whom one or both sphenoid sinuses are closed. Those with patent sinuses appear to run a milder course, though in a series of cases in which all had pus in the sphenoid, opening of this sinus did not result in much improvement. According to Thomas, Fleming and Lundi,[13] there is always a primary invasion of streptococcus and these are always obtained from first cultures. It is also worth noting that while the disease may have three distinct stages—catarrhal, septicaemic and meningeal—it may stop at either of the first two, and the case may only present symptoms of pharyngitis and laryngitis, or it may go on to rather severe influenza symptoms. The pharynx and pillars in these cases are deep red, the veins prominent, and the uvula generally edematous.

In regard to the treatment of meningitis of nasal origin shall we operate in the presence of symptoms which lead us to believe the meninges are threatened (meningismus) or involved? There is some difference of opinion as to this. We know that recoveries have followed proper surgical drainage in meningitis of aural origin. It seems logical then to believe that, provided we operate fully and completely in such cases, we should look for some cures, as the conditions are analogous. I believe with Luc [14] that half interference is worse than none in such cases. He quotes, in his long monograph on the subject, several cases in which all the sinuses involved were operated, except the antrum, and blames the fatal results on failure to do this at the same time. Given a case of meningitis, not epidemic, due to infection within the nose, I would advocate a thorough removal of all diseased structures. Where the path of infection to the dura is to be seen, the bone should be removed in all directions until healthy dura is reached.

Spinal punctures give relief of headache in all forms of meningitis, and greatly hasten the disappearance of the serous form, which, however, always recovers whatever treatment or lack of treatment is used.

Antimeningococcic serums have been disappointing, but vaccines seem to have been distinctly an aid where used.[15] Soamin intravenously [16] and collargol [14] have been successful in some cases, apparently.

As the case stands at present, we can do more to prevent than to cure the disease. When operation for infection is indicated, let it be thorough. Avoid procedures which have been known to cause involvement of the meninges, such as electro-cauterization of the middle turbinates, opening up the channels of the diploë in the posterior wall of the frontal sinus by too vigorous curetting at operation.[17] Clean, skilful surgery resulting in thorough drainage is our chief reliance at the present time, both as a preventive and as, I believe, a therapeutic measure.

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ECTOPIC PREGNANCY WITH REPORT OF CASE.

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The record of ectopic gestations in one of the leading eastern hospitals over a period of ten years, gives the astonishing information that less than 60% of these cases were diagnosed as such previous to operation. The mortality in unoperated cases is 68.8%, while in cases recognized early and promptly operated it is only 5%.

Here indeed is an opportunity to wipe out an unnecessary waste of human life, and it is the duty of every general practitioner (for it is to him that these cases first come for aid) to speed up in the early diagnosis of this condition and by prompt surgical measures save these patients from an untimely end.

Given the history in a case similar to the one recited below and any medical student ought to make the diagnosis, and yet in the minds of the profession there is much doubt and fear regarding this situation. Even among the teachers in the medical colleges one hears the subject lectured upon in such a solemn and fearful manner that he is apt to believe that he is on holy ground when approaching such a case, and it is but right and proper that he advance with fear and trembling. The readiness and certainty in the diagnosis of my first cases of extra-uterine pregnancies disillusioned me regarding the difficulty and exploded the false belief that this is a condition to be recognized only by the chosen few within the doors of large hospitals. The condition is not so rare but that it may happen in the practice of every doctor. That it is not recognized or at least suspected is in a measure due to failure of physicians to attach the importance deserved by the slight and transitory symptoms of the incipiency. In this respect the patients themselves commonly ignore the warning of what to them seems relatively an unimportant